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NEWS 25 NOV 15 Derwent Indian patent publication number format enhanced

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      15 MR16-1
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     3947 PMS
    47190 PM
        (PM OR PMS)
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     734 PM-1
        (PM(W)1)
     546 MRA
     63 MRAS
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        (MRA OR MRAS)
    132596 IL
     1570 ILS
    133636 IL
        (IL OR ILS)
   3998465 6
    32229 IL-6
        (IL(W)6)
   168956 INTERLEUKIN
    6421 INTERLEUKINS
   171012 INTERLEUKIN
        (INTERLEUKIN OR INTERLEUKINS)
   3998465 6
    40551 INTERLEUKIN 6
        (INTERLEUKIN(W)6)
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=> duplicate remove ENTER L# LIST OR (END):L1 PROCESSING COMPLETED FOR L1 76 DUPLICATE REMOVE L1 (0 DUPLICATES REMOVED) L2

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L3 76 S L2 2750 MESOTHELIOMA **580 MESOTHELIOMAS** 2909 MESOTHELIOMA

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1 L3 AND MESOTHELIOMA L4

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AN 2006:887900 CAPLUS

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TI Interleukin-6 induces both cell growth and VEGF production in malignant mesotheliomas

AU Adachi, Yasuo; Aoki, Chieko; Yoshio-Hoshino, Naoko; Takayama, Koichi; Curiel, David T.; Nishimoto, Norihiro

CS Laboratory of Immune Regulation, Graduate School of Frontier Biosciences, Osaka University, Osaka, Japan

SO International Journal of Cancer (2006), 119(6), 1303-1311 CODEN: IJCNAW; ISSN: 0020-7136

PB Wiley-Liss, Inc.

DT Journal

LA English

AB Malignant mesothelioma (MM), an incurable tumor, is reportedly an interleukin-6 (IL-6) secreting

tumor. The pathol. significance of IL-6

overexpression in this tumor, however, has remained unclear. We

investigated the biol. functions of IL-6 in

mesotheliomas. Five mesothelioma cell lines were

analyzed for IL-6 prodn. and IL-6

receptor (IL-6R) expression. Of them, 2 produced high levels of

IL-6, 2 produced intermediate levels and 1 cell line

showed no secretion. All mesothelioma cell lines used in this

study expressed very small amts. of IL-6R mRNA. We compensated for this

low level of IL-6R expression in mesotheliomas by adding

recombinant sol. IL-6R (sIL-6R) to mediate the IL-6

signal. IL-6 together with sIL-6R was found to

promote cell growth of H2052 and H226 MMs classified as high-level

IL-6 producers in a dose-dependent manner. Moreover, a humanized anti-IL-6R antibody (MRA) capable of blocking IL-6 signaling suppressed the cell growth of mesotheliomas induced by IL-6/sIL-6R. These findings demonstrate that IL-6 serves as an autocrine growth factor in the development of mesothelioma. In addn., IL-6/sIL-6R stimulation increased the expression of vascular endothelial growth factor (VEGF) in 4 out of 5 cell lines, and this induction was inhibited by MRA treatment. The involvement of the signal transducer and activator of transcription 3 (STAT3) pathway in both cell growth and VEGF induction by IL-6/sIL-6R was verified by dominant neg. STAT3 transduction combined with adenovirus gene-delivery methods. Although IL-6 induces VEGF through the JAK2/STAT3 pathway, anti-VEGF antibody could not inhibit the IL-6-induced cell growth obsd. in H2052 and H226. We concluded that IL-6-dependent growth does not occur via VEGF induction. These results suggest that treatment with anti-IL-6R antibody may constitute a potential mol. targeting therapy for MMs. RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD

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=> s L1 and mesothelium
2872 MESOTHELIUM
1 MESOTHELIUMS
19 MESOTHELIA
2882 MESOTHELIUM
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L5 1 L1 AND MESOTHELIUM

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AN 2006:887900 CAPLUS

DN 145:246150

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Γ	L12	L11 and (antagonist or inhibitor)	8				
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	L9	L8 and antibody	15				
	L8	L6 and angiogenesis and APJ	17				
	L7	L6 and angiogenesis	58				
	L6	((apln or apel or (agtrl1 ligand) or apelin) and (antagonist or inhibitor))	247				
	L5	(apln or apel or (agtrl1 ligand) or apelin) and (antagonist or inhibitor)	247				
	L4	L3 and (anti-apelin)	3				
	L3	L2 and (antagonist or inhibitor)	58				
	L2	L1 and angiogenesis	65				
	L1	(apln or apel or (agtrl1 ligand) or apelin)	4038				

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		arch apln or apel or (agtrl1 ligand) and giogenesis	13:19:53	3
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